(0.25 (95% CI 0.49 to 0.008) but not in the healthy participants. TNF-α, IL-17 and CRP were not significantly increased with exercise in either group. No significant differences were found between groups for the change (pre- to post-walk) in any inflammatory markers (CRP: p=0.07; IL-6: p=0.51; TNF-α: p=0.22; IL-17: p=0.44).

Abstract S30 Table 1 Baseline and post-walk systemic inflammatory mediators in patients with Chronic Obstructive Pulmonary Disease (COPD) and healthy comparators

Marker	Rest concentration	Post walk concentration	p Value
COPD (n=16)			
CRP (mg/l)	3.77 (1.67 to 8.49)	4.75 (1.99 to 11.08)	0.56
IL-6 (pg/ml)	2.80 (2.00 to 3.25)	2.95* (2.03 to 3.63)	0.04
TNF-α (pg/ml)	8.25 (5.80 to 10.13)	7.10 (4.95 to 10.03)	0.64
IL-17 (pg/ml)	52.52 (35.92 to 82.61)	55.16 (47.85 to 79.17)	0.62
Healthy (n=16)			
CRP (mg/l)	1.15 (0.61 to 2.60)	1.60 (0.68 to 2.60)	0.72
IL-6 (pg/ml)	2.70 (2.00 to 3.00)	2.00 (2.00 to 2.95)	0.73
TNF-α (pg/ml)	5.15 (4.00 to 9.18)	7.05 (4.00 to 9.08)	0.64
IL-17 (pg/ml)	65.50 (52.20 to 82.43)	70.40 (57.78 to 88.28)	0.31

*p<0.05 compared with rest concentration in the COPD group; all baseline concentrations (between groups) and other pre-post changes (within groups) were not significantly different (p>0.05).
COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein, IL-6, Interleukin-6, TNF-α, IL-17, Interleukin-17; mg/l, milligrammes per Litre; pg/ml, picogrammes per millilitre.

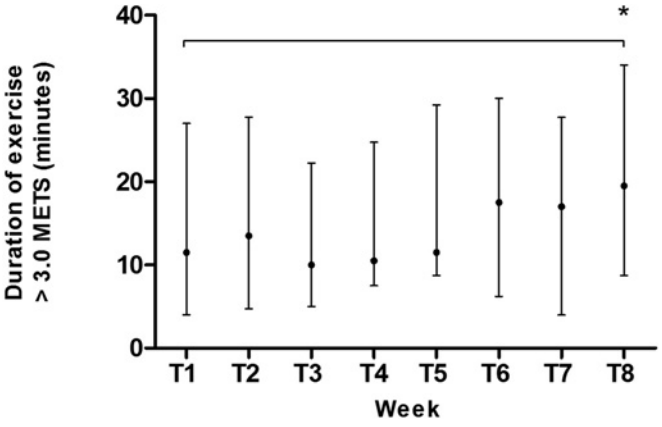
Conclusion Despite a significant increase in IL-6, the magnitude of the systemic inflammatory response to matched absolute workloads in COPD patients is not greater than in healthy comparators.

S31 ENERGY EXPENDITURE AND PHYSICAL ACTIVITY LEVELS DURING AN 8-WEEK PULMONARY REHABILITATION PROGRAMME

doi:10.1136/thoraxjnl-2011-201054b.31

J L Canavan, K A Ingram, R P Fowler, A L Clark, P Marns, M S Patel, S S Kon, W D-C Man. *Harefield Pulmonary Rehabilitation Team and Respiratory Biomedical Research Unit, Royal Brompton & Harefield NHS Foundation Trust, Harefield, Middlesex, UK*

Introduction The IMPRESS standards for pulmonary rehabilitation (PR) recommend that programmes should include two supervised exercise sessions per week for at least 4 weeks, and written prescriptions of exercise training with evidence of progress reported in training diaries. However, subjective self-reported assessment is associated with bias, and may not accurately represent actual exercise intensity and duration. Patients require familiarisation with equipment and training regimes during a PR programme. We hypothesised that active energy expenditure and time spent in at least moderate physical activity, measured objectively with a validated activity monitor (SenseWear armband—SWA), would show no significant increase within the first 4 weeks of a PR programme.
Method 34 COPD patients (17M: 17F), starting an 8-week outpatient PR programme consisting of two supervised exercise sessions per week, consented to wearing SWA for one entire exercise training session each week for the whole PR programme (T1—T8). Output from the SWA includes active energy expenditure (AEE) and time spent in at least moderate intensity physical activity (PA time) that is, >3.0 METS. AEE and PA time recorded at T1, T4 and T8 were evaluated using Friedman tests. Incremental shuttle walk (ISW) and COPD Assessment Test (CAT) were measured before (T0) and after (T9) PR. Differences in pre to post-outcome measures were assessed using paired t tests.
Results Results are presented as median (25th, 75th percentile). There was no significant difference in PA time or AEE between T1 (11.5 (4.0 to 27.0) min; 43.5 (19.5 to 124.3) Kcal) and T4 (10.5 (7.5 to 24.8); 48.5 (32.3 to 106.0) Kcal, p>0.05) despite progress documented in training diaries. PA time significantly increased from T1 to T8 (19.5 (8.8 to 34.0) min, p=0.02), as did AEE (92.0 (37.0 to 146.3) Kcal, p=0.006). Following PR there was also a significant improvement in ISW (52.1 (95% CI 29.2 to 75.1) m, p<0.001) and CAT score (−3.0 (95% CI 0.3 to 5.8) p=0.03).
Conclusion 4-week PR programmes may be insufficient in duration for patients to become familiarised with equipment and exercise regimes.



Abstract S31 Figure 1 PA time during pulmonary rehabilitation classes over an 8-week outpatient programme. Data presented as median (IQR) *p<0.05 significant difference compared to week 1.

Novel mechanisms driving airway inflammation in asthma

S32 LOSS/INHIBITION OF THE aVb5 INTEGRIN REDUCES ALLERGEN-INDUCED INCREASES IN AIRWAY SMOOTH MUSCLE MASS IN IN VIVO MODELS OF ASTHMA

doi:10.1136/thoraxjnl-2011-201054b.32

[1]A L Tatler, [1]A E John, [1]L Jolly, [1]A Habgood, [1]J Porte, [1]A J Knox, [2]X Huang, [2]D Sheppard, [1]G Jenkins. *[1]University of Nottingham, Nottingham, UK; [2]University of California San Francisco, San Francisco, USA*

Airway remodelling is a common feature of severe asthma. Transforming growth factor-ß (TGF-ß) is a pro-fibrotic, pleiotropic cytokine implicated in airway remodelling. TGF-ß is sequestered in the extracellular matrix as a latent complex and requires activation to function. We have previously shown that contraction agonists cause aVß5-mediated TGF-ß activation by human airway smooth muscle cells. The study aims were to investigate the role of the aVß5integrin in airway remodelling in vivo using two distinct mouse models of asthma. A blocking antibody directed against the aVß5 was used in the ovalbumin (OVA) model of asthma. Mice were sensitised with OVA/Alum on days 0 and 12, then challenged byoropharyngeal administration of OVA 10 times over 2 weeks. The anti-aVß5antibody or an isotype matched control antibody was administered for the duration of the OVA challenges. The second invivo model utilised *itgb5*$^{-/-}$mice. *Aspergillous fumigatus* antigen preparation was administered intra-nasally (10 µg/mice) to *itgb5*$^{-/-}$ and wild type controls 9 times over a 21-day period. a-Smooth muscle actin (a-SMA) was quantified in lung sections from both studies by immunofluorescence. Murine airway smooth muscle cells express aVß5 integrin and can activate TGF-ß in vivo in response to allergen challenge as measured by αvβ5 and phospho-Smad2 immunostaining. Treatment with both OVA and *Asp.f* resulted in an increase in a-SMA staining around the smaller airways. The aVß5blocking antibody significantly reduced a-SMA staining compared with the